MODELING OF EQUIVALENT NONLINEAR RESISTANCE AND CAPACITANCE OF ACTIVE COCHLEA

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Abstract - **This paper presents an ear model that combines the classical transmission-line cochlear model, the active cochlear partition model, and the active model of a single outer hair cell (OHC). This model can successfully generate "tall and broad" basilar membrane response and otoacoustic emissions. Result show that, owning to the OHC activities, the active cochlear partition has an equivalent resistance always lower than the resistance of a passive cochlear partition; and that the active cochlear partition has an equivalent capacitance always higher than the capacitance of a passive cochlear partition.**

I. INTRODUCTION

It has widely been accepted that outer hair cells (OHCs) underlie the amplifier mechanism, especially after the findings of Brownell et al. showing that OHC could twitch when stimulated electrically [1]. Over the past years, many experimental data on single OHC have been accumulated. Zheng et al. recently reported that his group had already found and cloned successfully the gene responsible for the active force generation from the OHC membrane [2].

Despite of rich experiments on signal OHC, the mechanism of active force in generating the high sensitivity and frequency selectivity of basilar membrane (BM) and otoacoustic emissions (OAEs) is still unknown. For the lack of the knowledge on the active mechanism, it is difficult to develop an efficient signal processing method that can mimic the function of a health cochlea in processing speech. Modeling could provide an aid in understanding the mechanisms responsible for the experimental observations and the behaviors of cochlea.

Most of the models developed previously for active cochlea were based on the studies of OAEs, partially because OAEs can be objectively measured at ear canal and viewed as an observation of OHC activities. Zweig, Talmadge, et al. developed a model with a nonlinear resistance and two (fast and slow) stiffness feedbacks based on OAEs [3-5], and demonstrated that the model could successfully generate the "tall and broad" BM response, transient evoked otoacoustic emissions (TEOAEs) and distortion production otoacoustic emissions (DPOAEs) [5-7]. Hubbard and Mountain used a two-transmission-line-model to simulate the "tall and broad" BM response and TEOAEs [8]. Hubbard concluded that both the nonlinear resistance and the nonlinear capacitance must be used to obtain reasonable comparisons with electrically evoked OAE data [9]. Egbert de Boer reviewed several forms of locally active models of the cochlea in the literature [10]. In those models, the usage is made of a secondary resonance or a place-dependent delay to achieve activity over a limited part of the length of the basilar membrane, and OHCs are postulated as the sources of energy production [10]. Egbert de Boer showed that all these models could be reduced to a standard form, in which the activity of OHC was represented by a nonlinear resistance and a nonlinear capacitance. The nonlinear resistance and capacitance are also often used in the signal cellular model of OHC [11]. All these evidences show that the effect of the OHC on BM can be expressed by a proper nonlinear function of resistance and capacitance. However, the functions of the nonlinear resistance and capacitance used in these models were different, and were based only on the qualitative description of the active forces that had limited foundation of the OHC experimental data.

In order to determine the formulas of the nonlinear resistance and capacitance, we will present an OAE model, which includes the models of ear canal, middle ear, and cochlea. Based on this model, we will derive a mathematical formula in terms of nonlinear resistance and capacitance for understanding the active mechanism of cochlear. Finally in this paper, we will show that with these nonlinear resistance and capacitance, the model could successfully generate the "tall and broad" BM response and OAE signals, especially TEOAEs and DPOAEs.

II. EAR MODEL

Since OAEs are generated in cochlea and recorded in the ear canal, the OAE model proposed in this thesis includes all the parts of the ear canal, the middle ear, and the cochlea. The models of ear canal and of middle ear are adopted from the model proposed by Giguere [12] and later adopted as the OAE model by Zheng [13].

Before modelling the active cochlea, it was assumed that all the OHCs in a small enough segment work in a locked-phase. And thus, these OHCs were defined as an OHC-group that has the same structures, performance, and function. The supportive evidence of this assumption is coming from the anatomy of the OHC, which shows that the stereocilia are composed of bundles of actin filaments that are extensively cross-linked, and the cuticular plate, into which the stereocilia rootlets are planted, is similarly made up of a mosaic of cross-link actin filaments [14]. These links between the stereocilia allow a quick opening synchronized for all stereocilia when they are displaced. The second assumption is that the open/close status of the ionic channel, which leads to the change of OHC potential, is related to the BM displacement and its velocity. This assumption is a general one. The third assumption of this active cochlear model was that the force generated by the OHC motors is dependent on the OHC membrane potential. This assumption is supported by lots of the experimental results on signal OHC [15-17].

Based on these three assumptions, the resultant model of active cochlea is shown in Fig. 1. In Fig. 1 (a), each block represents a local model of cochlear partition (CP) on the BM, which is shown in details in Fig. 1 (b). In Fig. 1 (a), L_{sn} represents the acoustic mass of the scalae vestibuli and tympani fluids; and L_T represents the acoustic mass of cochlear fluid from the last BM segment to the helicotrema. In Fig. 1 (b), U represents the mechanical force generated by the travelling wave in the cochlea; and R_n, L_n, and C_n represent the passive acoustic resistance, mass and compliance of CP in BM, respectively. The

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values of all these passive parameters are adopted from the model of Giguere [12]. Based on the third assumption, the voltage source, $g_1 v_m$, in the left branch of Fig. 1 (b) represents the active force generated by OHC-group, whose model is shown in the right hand of Fig. 1 (b), where R_a represents the resistance in endolymoh-facing cellular apex of the OHC-group, and R_m and C_m consist of the perilymph-facing basolateral membrane impedance in the OHC-group. The usage of the model of a signal OHC to describe the model OHC-group is based on the first assumption. In the right block of Fig. 1 (b), the voltage source, $g_{21} v_n$, and the current source, $g_{22} i_n$, represent the effects of BM displacement and the velocity of the BM displacement on the ionic channel status, respectively. These two dependent sources are introduced based on the second assumption. It is clear that the model proposed here is a combination result of the active system cochlear model, the model of active cochlear partition, and the cellular model of OHC.

The values of all these parameters are difficult to identify. According to the reports [15-17], the factor g_1<0 and is in the order of –5 to –7; the factor g_2>0 and is in the order of 4 to 6; R_a, R_m are larger than 0 and in the order of 4 to 5 in the unit of ohm; and C_m are larger than 0 and in the order of –11 in the unit of farad.

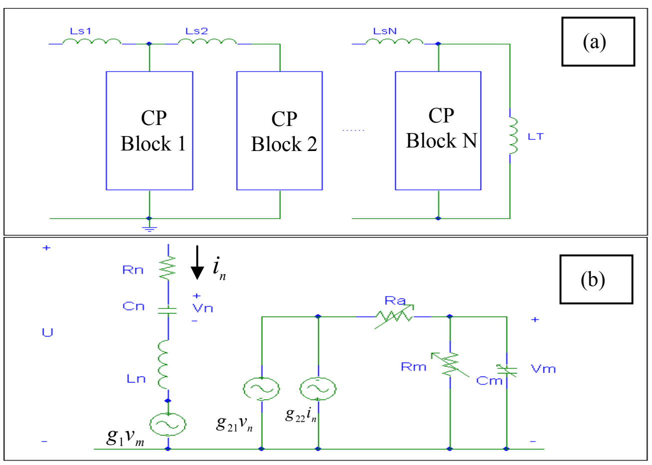

Fig. 1. The model of (a) the active cochlea and (b) the active cochlear partition.

Given R_a, R_m, and C_m to be constants in a short enough time, a mathematical model with nonlinear equivalent resistance and capacitance can be derived from above active electronic cochlear model. The resultant mathematical model is expressed by

$$L_n \frac{d^2 v_n}{dt^2} + R_{eq} \frac{dv_n}{dt} + \frac{1}{C_{eq}} v_n = U \,, \qquad (1)$$

where

$$C_{eq} = [1 + \frac{g_1 g_{21} C_m}{R_a + R_m}(1 - \exp(-\frac{R_a + R_m}{R_a R_m C_m} t))]^{-1} C_n \,, \qquad (2)$$

and

$$R_{eq} = [1 + \frac{g_1 g_{22} C_m}{R_n (R_a + R_m)}(1 - \exp(-\frac{R_a + R_m}{R_a R_m C_m} t))] R_n \,. \qquad (3)$$

Further derivation shows that

$$C_{eq} = [1 + \Gamma_c (v_m, \frac{dv_m}{dt}, \cdots)] C_n \,, \qquad (4)$$

and

$$R_{eq} = [1 - \Gamma_R (v_m, \frac{dv_m}{dt}, \cdots)] R_n \,. \qquad (5)$$

where $\Gamma_c(\bullet) > 0$ and $\Gamma_R(\bullet) > 0$. It is clear from Eqs. (4) and (5) that the equivalent resistance is always lower than the passive resistance, and the equivalent capacitance is always larger than the passive capacitance.

III. RESULTS

Computer simulations were conducted to verify the mathematical model. Under the constraints of Eqs. (4) and (5), we approximately set the equivalent resistance as

$$R_{eq} = (1 - G_1 \frac{d_{1/2}}{d_{1/2} + |k_n v_n|}) R_n \,, \qquad (6)$$

where $k_n = \frac{C_n}{b(x_n)\Delta x}$; $b(x_n)$ is the width of the nth segment; $d_{1/2}$ is a constant equal to the BM displacement at the half-saturation point of the nonlinearity; G_1 is the active gain factor, whose value is related to the activity of the corresponding OHC-group; and the equivalent capacitance as

$$C_{eq}(t+\tau) = (1 + G_2 |\frac{dv_I(t)}{dt}|)^2 C_n \,, \qquad (7)$$

where G_2 is the active gain factor whose value is also related to the activity of the corresponding OHC-group; and v_I is the inertial item that is equal to the mean value of $v_n(t)$, $v_n(t-\tau)$, and $v_n(t-2\tau)$, and τ is the calculation step. The averaging of v_n over the short period time is used to simulate the effect of low pass filter induced by OHC.

When simulating, the active factor of the resistance G_1 was set as 0.95 plus a normally distributed random value ranged from –0.05 to 0.05 to simulate the inhomogeneous property of the cochlea. And no parameter identification was performed. The simulated OAE results were then compared with the signals recorded from the normal human ear. The TEOAEs and DPOAEs were recorded by the ILO88 and ILO92 systems in a quite room without soundproof. All the subjects were in the age from 22 to 27 year-old and had normal hearing.

Fig. 2 shows the BM response simulated from the model proposed in this paper. When doing simulation, the parameter G_2 was set at $(4 + C) \times 10^4$, where C is a normally distributed random number with values in the range of –0.15 to 0.15. In Fig. 2, the dotted line, dashdot line, dashed line, and the solid line are the results of N=128, 256, 512, and 1024, respectively. It can be seen from Fig. 2 that the dashed line and the solid line for N=512 and 1024 are almost overlapped completely, which implies that when N=512, the solution of the nonlinear function is stable. Therefore, in the later computation simulations N was always set as 512. It is also clear from Fig. 2 that the "tip-to-tail" ratio of simulated BM response is more than 30 dB SPL and as broad as several kHz. These characteristics are consistent with the findings of the physiological experiments by Ruggero [18].

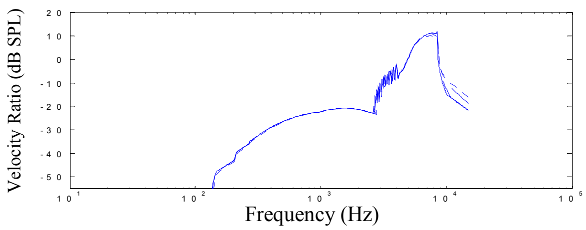

Fig. 2. The simulated BM response.

Figs. 3 and 4 show a typical TEOAE recorded from a normal human ear and the results of computer model, respectively. The signals in the time domain, their corresponding magnitude responses and the phase responses in the frequency domain are shown in the subplots (a), (b), and (c), respectively in Figs. 3 and 4. Both the recorded TEOAE and the simulation results were stimulated by a click stimulus as follows:

$$u_{in}(t) = \begin{cases} A\sin(2\pi \times 12.5kHz \times t), & 0 \le t < 80\,\mu s \\ 0, & \text{others} \end{cases}, \qquad (8)$$

where A=80 dB SPL. When doing the simulation on ourmodel, the parameter G_2 was set as $(8+C)\times 10^5$, where C is the same as that used for the study of BM responses. It is clear that the recorded TEOAE and the simulated TEOAE have the similar pattern in both time domain and frequency domain.

Figs. 5 and 6 show the recorded DPOAE and the simulated DPOAE, respectively. For recording and simulating the DPOAE, the stimulus levels are set at 70 dB, and the frequencies of the two primaries (f_1 and f_2) are set as 1000 Hz and 1280 Hz, respectively. For the model simulation, the parameter G_2 was set as $(4+C)\times 10^4$, where C is again the same normally distributed random number as that for BM responses. It can be clearly seen from Figs. 5 and 6, that in both the recorded DPOAE and simulated DPOAE signals, the cubic distortion product (CDP) at $2f_1$-f_2 is clear, and the magnitudes of the CDP are about 60 dB SPL less than the magnitudes of the two primaries. These properties are consistent with those reported previously [19].

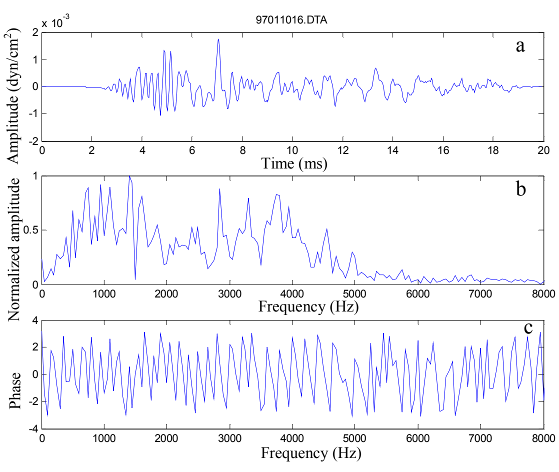

Fig. 3. The TEOAE signal recorded on the ear of a human subject with normal hearing.

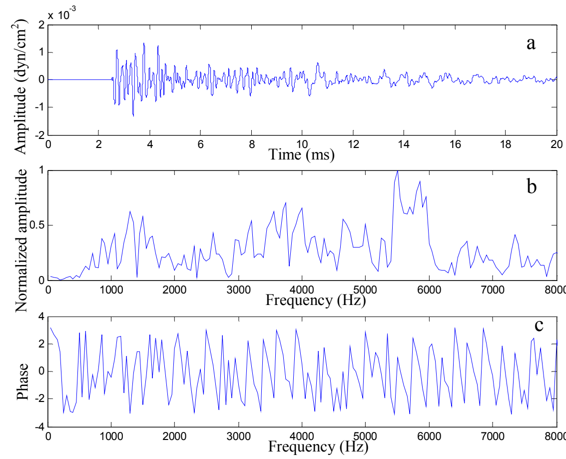

Fig. 4. The TEOAE signal generated by the mathematical model.

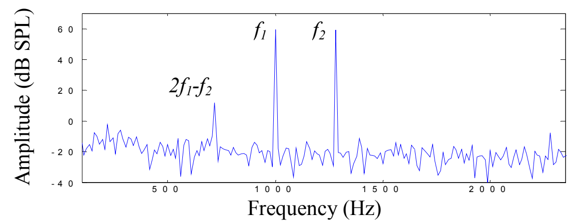

Fig. 5. The recorded DPOAE signal.

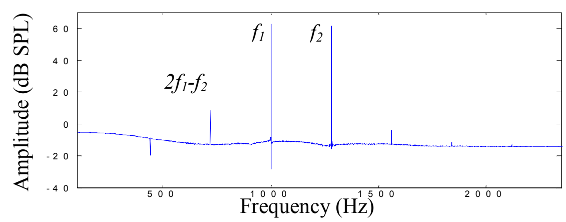

Fig. 6. The simulated DPOAE using the mathematical model.

IV. DISCUSSION AND CONCLUSION

In this paper, an electronic one-dimensional model of the active ear including the ear canal, the middle, and the cochlea for the OAEs has been developed based on the classical passive cochlear model, the active cochlear model, the active cochlear partition model, and the signal OHC cellular model. The parameters in this model have clear physiological meanings. Based on this electronic model, we have derived the mathematical model in which the active mechanism of the OHC function was described by two nonlinear functions of resistance and capacitance, which reflect the effects of OHC.

The computer simulations have been conducted to verify the mathematical model. It has been shown that this model could successfully generate the "tall-and-broad" property of BM response, which was proved to be necessary and sufficient for OAE generation. It has also been shown that this model could successfully generate the TEOAE and the DPOAE.

This work actually supports the previous models proposed by Zweig and Talmadge, and by Hubbard on the following grounds. First, all these models are based on the classical passive one-dimensional transmission line cochlear model. Second, both the models of Zweig and of Hubbard, and the model proposed in this paper introduce the nonlinear resistance and capacitance. Third, the OHC in all these models works like a low pass filter.

Besides all these consistency, the characteristics of the model proposed here are that: 1) the active mechanism of OHC was described clearly by two nonlinear functions that represent the OHC resistance and capacitance functions, respectively; and 2) both electronic model and mathematical model are used to describe the active cochlea. The electronic model has clear foundation of physiology, and the derived mathematical model has clear formula of the active mechanism. Based on this model, it has been found that the equivalent resistance is always lower than the passive resistance, and that the equivalent capacitance is always larger than the passive capacitance.

Although Eqs. (6) and (7) are proved to be useful to describe the BM response and to generate OAEs, the active gain factor G_2 has to be set as different values when the stimuli are at different levels. Nevertheless, this work is promising to identify the active mechanisms of cochlea with the suppport of experimental data on single OHC.

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